

5. Schlesinger N. Dietary factors and hyperuricaemia. *Curr Pharm Des.* 2005;11:4133–8.

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Reply to the letter "Long term effects of nocturnal hypoxia and urinary uric acid excretion: How much linked to COPD and OSAS?"



Dear Editor,

We have read the letter titled "Long term effects of nocturnal hypoxia and urinary uric acid excretion: How much linked to COPD and OSAS?" regarding our manuscript 'Urinary uric acid excretion as an indicator of severe hypoxia and mortality in patients with obstructive sleep apnea and chronic obstructive pulmonary disease'. First, we would like to thank Dr Gomes and colleagues for their interest in our study. We are grateful for the insightful comments to our article and are happy to respond to their comments as follows.

1. First of all COPD and OSA have different mechanisms for hypoxemia as mentioned. Furthermore intermittent and chronic hypoxemia has different effect on diseases too. It would be very useful to compare these results on uric acid levels in both diseases, but unfortunately as we mentioned in our manuscript we did not have enough participants to make this sub-group analysis.
2. As the author mentioned there is no universal definition in the grading of nocturnal intermittent hypoxemia.¹ It is neither wrong nor faultless to use >10% or >30% of sleep time.²⁻³ It would be more useful to use both criteria and compare the results in a large group of patients.
3. All the COPD patients were taking maximal pharmacological and non-pharmacological therapy according to the GOLD guidelines. But it is a very nice comment to

make further studies with new GOLD combined assessment strategy.

4. Hypertension was the case for approximately 15% of our patients, which could decrease UA excretion. However, the analysis did not reveal a significant difference between hypertensive and normotensive patients.

We believe that further clinical trials investigating the effect of nocturnal progressive and intermittent hypoxemia on COPD and OSA patients are needed to confirm our results.

Conflicts of interest

The author has no conflicts of interest to declare.

References

1. Neubauer J. Physiological and genomic consequences of intermittent hypoxia. Invited review: Physiological and pathophysiological responses to intermittent hypoxia. *J. Appl. Physiol.* 2001;90:1593–9.
2. American Academy of Sleep Medicine. International Classification of Sleep Disorders: Diagnostic and Coding Manuel. ed 2 Westchester, IL: American Academy of Sleep Medicine; 2005.
3. Chiang AA. Obstructive sleep apnea and chronic intermittent hypoxia: a review. *Chin J Physiol.* 2006;49(5):234–43.

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